

CONTRACT REPORT NO. 05T84-00460

EXAMINATION OF THE IMPACT OF POLYNUCLEAR
AROMATIC HYDROCARBON EXPOSURE IN UNDERGROUND MINES

Contract No. 24ST.23440-4-9142

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MICROMEDIA
JUL 18 1989

INDEX

	Page
Executive Summary	1
1. Introduction	3
2. The Development of Knowledge Concerning Lung Cancer and Airborne Pollution	4
2.1 Major Review Documents	17
3. Biological Evaluation of the Carcinogenic Potential of Air Pollution - The Ames Test	31
4. Commentary on Review Documents	34
5. Polynuclear Aromatic Hydrocarbons in Ontario Mines	36
5.1 Introduction	36
5.2 Sampling Methods	36
5.3 Analytical Methods	39
5.4 Exposure Estimates	40
6. An Approach to the Evaluation of the Health Effects of PAH's in Ontario Mines	43
References	54
Table #2	i-ii
Table #3	i
Table #4	i-ii
Table #5	i-ii

Executive Summary

Only limited epidemiological evidence is available on the risk of lung cancer associated with the use of underground diesel equipment. The purpose of the present report has been to examine the possibility of making inferences about the risk by considering two sources of information. On the one hand Westaway and Faulds (1982) have reported the results of measurements of PAH's in the atmosphere of Ontario mines when diesel equipment was in use. On the other hand extensive epidemiological information is available which relates the risk of lung cancer in urban populations exposed to PAH's in the atmosphere.

The use of a single PAH (Benzo[a]pyrene) has been widely adopted as the reference PAH in air pollution studies. We believe that very great caution must be exercised in using this PAH as an indicator of risk in the mining environment and of relying on data obtained from urban populations for quantifying the potential risk. Indeed, the use of Benzo[a]pyrene as an indicator of risk in city air is difficult to justify. The same reservations apply to the interpretations of biological tests (the Ames test).

Because of the sampling strategy chosen by Westaway and Faulds (1982) it was not easy to estimate the full shift exposure to miners. An approach to this was chosen which made the maximum use of all data. From such calculations it was possible

to show that the PAH levels to which miners were exposed was fairly similar to that found in current city atmospheres. With particular attention to the reservations specified above this would suggest that a risk of lung cancer is unlikely to be greater than that associated with current city air pollution. In no circumstances can this be interpreted as claiming that there is no risk.

Since the PAH's and related compounds derived from diesel machines are positive on Ames testing there is a priori reasoning to suggest that they must be assessed as possible aetiological agents causing lung cancer. For this reason all attempts must be made to develop technology that will lower whatever levels are current in mining operations. We believe that filtration equipment is likely to be beneficial in this respect. Areas where research is needed are discussed. Despite the limitations of epidemiological surveys there are cogent reasons for suggesting that plans should be made at the present time so that information will be available in subsequent years.

I INTRODUCTION

The purpose of this review is to assess the health implications of the airborne concentrations of potentially carcinogenic substances in the air of underground mines in Ontario as reported by Westaway and Faulds (1982). The issues raised are very similar to those which were relevant in the early part of the century when the foundations of experimental work on cancer were first established. For this reason a historical review will first be presented to illustrate the way in which the methodologies of epidemiological research have developed in the field of air pollution and its effect on human health. The target organ is the lung and the focus will be on the etiological determinants of carcinoma of the bronchus. The basic questions concern the extent to which this can be attributed to one or more substances in the atmosphere of cities or in the workplace and the validity of estimating risks in one set of exposure conditions from information obtained in other situations. This review will be followed by a consideration of the sampling methods and analytical techniques used by Westaway and Faulds (1982) and from comparison of these with techniques used most often in sampling air pollution. Finally, an attempt will be made to evaluate the health implications of the Westaway and Faulds (1982) data in terms of information of pollution in the atmosphere and also in other workplaces.

2. THE DEVELOPMENT OF KNOWLEDGE CONCERNING LUNG CANCER AND AIRBORNE POLLUTION

Experimental work on the causes of human cancer developing from the observation that workers in certain occupations which were known to be associated with exposure to soot or to coal tar products had an increased risk of developing cancer of the skin. Percival Pott (1775). This was the first instance in which it appeared that a cause of cancer could be identified and resulted in extensive research in the early 1900s in an attempt to identify the substance or group of substances that might be the responsible agents. It is of some interest to note that even at this time it was claimed that workers in equivalent industries but in different countries appeared to be at substantially different risk. For example, skin cancer was common in coal tar pitch workers in Britain but this condition was claimed to be extremely rare in similar workers in France Woglom (1926). While such differences might be due to different work practices or to very different working conditions there was also a possibility that it could be due to some chemical difference associated with the industrial process. This is an important early example of the care that is required in transferring risk estimates from one industrial situation to another unless precautions are taken to standardize for exposure to the appropriate etiological agents.

These early observations stimulated an intensive search for the identification of the chemicals which were present in tar or soot and to experiments designed to induce cancer in animals by repeated painting of the skin with tar or soot. It was assumed that repeated skin contact by various chemicals was the causative mechanism of human skin cancer and it appeared logical to repeat the process in the experimental animal model.

Unfortunately it was not realized at the time that there are important differences in the susceptibility of animals to develop skin cancer. Research groups carried out extensive experiments using very large numbers of animals and consistently reported negative results. This paradox remained unexplained until Japanese workers had the good fortune to choose, by chance, a susceptible species and to continue painting the skin for the very prolonged period of time that is now known to be required. Yamagiwa and Itchikawa (1914) finally reported that skin cancer could be caused in mice by repeated painting with tar. This original paper has become one of the classic contributions in the field of cancer research. It rapidly became obvious that this was due to some specific chemical or substances in the tar and was not due to a simple irritant effect. This itself was an important contribution since tar painting caused chronic inflammatory changes and it was important to demonstrate that the persistent inflammation was not itself a cause of cancer. Many other substances such as alkalis or acids were well known to cause chronic irritation or inflammation but these

changes were not, in themselves, sufficient to cause malignant change. Following the discovery by Yamagiwa and Itchikawa that there was something specific about the ability of tar products to cause cancer the next step was the problem of identifying the particular chemicals. It is worth emphasizing that nobody at the time believed it likely that a single chemical was involved Woglom (1926), even though, as will be noted below, attention gradually became focused during the next few years on one substance.

The difficulty of sorting out the cancer agents in coal tar was due to the fact that it appeared to contain several hundred substances in various concentrations and only about 100 of these had been identified by (Woglom 1926). During the industrial production of coal tar the material was normally separated by distillation into 4 or 6 fractions of differing boiling points. The various research groups carefully tested each of these various fractions for carcinogenic potential by means of animal skin testing. The most important fraction appeared to be that which was distilled at temperatures greater than 200o C. A number of different compounds were identified, either because they were present in relatively high concentrations, or because they gave rise to specific chemical compounds which could be fairly easily analyzed. However, none of these appeared to cause cancer when tested on animals in the way that had been obtained with the parent tar. Increasingly it appeared that the agents responsible for cancer might be highly specific and might

be causing their effect despite being present in exceedingly low concentrations.

The analytical problems were formidable until Hieger (1930) at the Cancer Research Institute in London observed that certain tars and oils, in addition to being capable of causing skin cancer also showed intense fluorescence when exposed to ultraviolet light. Finally, Cook et al. (1933) isolated the fluorescent material from the distillate of coal tar and found that this was largely due to 1:2benzpyrene. They also found this chemical to be highly carcinogenic. They concluded that this chemical was certainly one of the potent substances in tar to which its carcinogenic properties could be attributed. The fortuitous properties of 1:2 benzpyrene which make it fluoresce in ultraviolet light and which also happen to account for its carcinogenic potential greatly assisted research and also to some extent account for the increasing attention that was subsequently given to this chemical. Since it was now known that coal tar when heated to an appropriate temperature produced carcinogens, many other materials were also heated to above 700o F. These included isoprene, acetylene, human skin and muscle and all of these were found to produce carcinogens but of unknown identity (Kennaway 1930).

Research interest at this time shifted to the study of synthetic carcinogens and the probability that other carcinogens were present in tar does not appear to have been followed up. It seems to have been assumed that 1:2 benzpyrene was the only

carcinogen in tar despite that fact that the response to tar painting in animals was very different from that due to the application of pure 1:2 benzpyrene. Berenblum and Schoental (1947) decided to study this problem and were able to show that tar from which all the 1:2 benzpyrene had been extracted still had strong carcinogenic potential. However, they were not able to identify the nature of the substances.

Epidemiological evidence of human cancer was beginning to focus on the rising death rate from lung cancer and Cooper (1955) first observed that there was a difference in the rates of lung cancer reported among city residents compared to those living in the country. Kennaway and Kennaway (1947) first suggested that this might be due to an etiologic factor contained in the smoke from burning coal fires. These observations referred to the United Kingdom at a time when the standard method of heating homes was the unrestricted burning of coal with the production of intense air pollution in cities. This proposal received support when Goulden and Tipler (1949) first identified 3:4 benzpyrene in extracts of domestic soot. Finally, Waller (1952) isolated 3:4 benzpyrene from samples of air pollution obtained by drawing ambient city air through filter papers. The concentrations ranged from 2-15 mg/100m³ of air and varied considerably with the atmospheric conditions. In general it was higher in winter and was positively correlated with the size of the town or city. In their reports Waller (1952) comment that they did not seek to identify other hydrocarbons in the air. It

was also of much interest that they were unable to identify 3:4 benzpyrene in tobacco smoke although this chemical and other carcinogens were later identified by Cooper and Lindsay (1955). The amount of 3:4 benzpyrene in tobacco smoke appeared to be about 4.0 ug in the total smoke collected from 500 cigarettes.

In 1955 Stocks and Campbell compared the urban/rural death rates for lung cancer in Britain and compared these with estimates of the airborne benzpyrene concentration. The urban/rural gradients for 3:4 benzpyrene concentration appeared to be about 10 - 1 although, as noted there were considerable variations according to the time of year. Since this ratio was fairly similar to that of the lung cancer death gradient they suggested that 3:4 benzpyrene might be the specific causative agent of lung cancer. They added there was no reason at the time to suspect any other particular compound although this possibility was certainly not excluded.

By 1960 Sawicki et al. were able to report on benzo[a]pyrene concentration in the air of a number of American cities. This chemical was selected as being a semi-quantitative index of the presence in air of other polynuclear hydrocarbons, some of which were known to be carcinogenic in animals and which were also suspected of producing cancer in man. Benzo[a]pyrene had also frequently been used in various animal experiments as an 'index' or measure of carcinogenic potential for comparison with other members of the polynuclear hydrocarbon family.

In discussing the validity of benzo[a]pyrene (BAP) as an

Index of carcinogenic potential In the air the authors stressed that at least 5 other potential carcinogens were known to be present in urban air although these were probably less carcinogenic than BAP. The list included benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i.]perylene and chrysene. Finally the authors noted that the intrapulmonary deposition and retention of benzpyrene and probably of other polynuclear hydrocarbons might be affected by the number, amount and size of particulates present in the air upon which the specific agents might be absorbed. In their estimation the urban/rural gradient for BAP concentrations was approximately 100 fold. This gradient is considerably greater than that of the corresponding lung cancer mortality gradient.

In a later report Sawicki et al. (1962) extended their study to include measurements of the concentration of a number of other polycyclic hydrocarbons which were thought to be carcinogenic. They found that there were fairly close correlations between at least seven hydrocarbons (Benzo(g,h,i)-Perylene, Benzo[a]pyrene, Benzo[k]fluoranthene, Pyrene, Perylene and Anthanthrene), such that any one could be used as a reasonable predictor of the concentration of the others. Coronene appeared to be an exception and its concentration did not correlate well with that of the other compounds. Some compounds (particularly BAP) appeared to be particularly stable in air while others such as pyrene were much less stable and were particularly susceptible to degradation in sunlight. The ratio

of pyrene to BAP may thus serve as an indicator of the interval since a particular pollutant atmosphere was first formed. There were also differences in the distribution of chemicals within the atmospheric pollution according to its source. Coronene and benzo[g,h,i] perylene were particularly characteristic of automobile exhaust and the ratio BAP/CO and BAP/BGHIP were suggested as possible indices of the extent to which air pollution was caused by motor traffic.

Meanwhile back in the U.K. Stocks and colleagues (Stocks and Campbell, 1955; Stocks, 1960; Stocks et al., 1961) had undertaken similar studies of the air in cities of various size. Smoke collected over a period of 12 months was extracted with cyclohexane from Whatmen No. 1 filter papers and was analyzed by spectrophotometry for 3,4 benzpyrene, 1,12 benzperylene, pyrene, fluoranthene, 1,2 benzpyrene, coronene and anthranene. Phenanthrene and anthracene were determined but the amounts were too small for accurate measurement. Special care was taken to eliminate interference between overlapping chromatographic fractions where this was possible. Average concentrations for each substance are given (concentrations were expressed as ug/1000m³) although the absolute weight of material collected was obtained by rather indirect means. The authors noted that the relevant proportions of the different hydrocarbons were very variable with the exceptions of coronene and anthracene which appeared to be fairly constant. In contrast to the work of Sawicki et al. (1962) they did not find that there was a good

correlation between the various compounds and that no single substance could be used as a reliable predictor of the concentrations of the other substances. They noted that correlations between different hydrocarbons might be affected by instability of certain individual substances due to evaporation or oxidation after collection on the filter paper. This lack of correlation in fact turned out to have considerable practical importance since it enabled them to compare the relationships between individual substances and lung cancer mortality. The closest correlation appeared to be with that of the airborne concentration of 3,4 benzpyrene and they also noted that this corresponded fairly closely with the relative potency of various substances which had been compared in animal experiments. The high point of the theory that lung cancer was due to air pollution may be identified in a paper published by Pybus in 1963. His arguments were based mainly on the proposition that the greatest mass or weight of chemicals known to produce cancer were produced by the burning of fossil fuels and the total weight of carcinogens produced by smoking cigarettes was very much less. This comparison led him to the erroneous conclusion that cigarettes were a less important cause of cancer than air pollution. Unfortunately his arithmetic did not make due allowance for the dilution of air pollution in the total environment. In actual practice this results in relatively low levels of ambient carcinogens in comparison with the highly concentrated amounts that are inhaled with each puff of cigarette

smoke.

Although these early experiments have found various levels of correlation between a number of chemicals it was increasingly clear that a complete analysis of the carcinogenic determinants of urban air was likely to be very complicated. For example, Sawicki et al. (1965) analyzed air from urban atmospheres and identified a number of new organic compounds which were known carcinogens including polynuclear aromatic hydrocarbons and polynuclear azoheterocyclic compounds. Co-carcinogens and even a number of substances which had an anti-carcinogenic effect were found. They concluded that no single substance could be accepted as a single indicator of the health effect of a given atmosphere. In parallel with these research studies there was rapidly increasing interest in the contribution of cigarette smoke to lung cancer in the community. In order to evaluate the effect of air pollution it was clearly important to make due allowance for cigarette smoke. Two studies of emigrants from the heavily polluted cities of Britain were influential in emphasizing the relative importance of early exposure to air pollution. Eastcott (1956) analyzed mortality in migrants to New Zealand and Dean (1959) in emigrants to South Africa. Both studies appeared to show that urban air pollution exposure in Britain during early life resulted in lung cancer rates many years later which could not be attributed to cigarette smoking. Even when standardized for cigarette smoking, the early years of life in Britain seemed to confer on the individual an increased tendency to develop lung

cancer which could not be due to air pollution in the new country. In an effort to elucidate the problem the large air pollution surveys in Britain were extended to include surveys of cigarette smoking in the exposed populations (Stocks 1966). The data from the U.K. and also from other countries demonstrated that air pollution and cigarette smoking were themselves closely correlated and that this made it extremely difficult to distinguish the effects of one from the other. This correlation does not imply that cigarette smoking causes air pollution but that populations who live in large industrialized cities also happen to be the very same populations who smoke heavily. It was also becoming evident that the urban rural gradients in lung cancer deaths which had been attributed to air pollution in the early studies appeared increasingly to reflect differences in smoking levels. Even allowing for these differences however there still appeared to be a component of the excess lung cancer risk which could be attributed to air pollution.

Analyses of this type consist in comparing the overall lung cancer rates in urban residents with those living in the country and at the same time making inferences from surveys which compare the mean rates of smoking in cities with similar average figures from the countryside. The problem with overall averages of this type is that they can easily conceal the real magnitude of an effect. For example it is necessary to compare lung cancer rates in smokers with those in non-smokers before the overwhelming effects of smoking are evident. In general the

relative risk for smokers is increased by a factor of about 9 and the risk is very much higher amongst heavy smokers. In the analyses by Stocks (1966) the urban/rural gradient was modest overall and the data did not permit inferences about the magnitude of the effects of air pollution in the absence of smoking.

Increasing interest in the effects of air pollution and of cigarette smoking stimulated large studies in a number of countries. Buell and Dunn (1967) and Hammond and Horn (1958) found that lung cancer deaths were about 25% higher in cities compared to rural areas amongst smokers but that the ratio was higher (about 3 to 4-fold) amongst non-smokers. In reviewing a number of studies Buell and Dunn (1967) noted that most had found an increased urban/rural lung cancer gradient but that at least two had failed to demonstrate an effect of air pollution in causing lung cancer amongst non-smokers. A number of methodological problems were identified and the most important of these was the difficulty in establishing an unbiased estimate of smoking levels. The authors also noted that there were likely to be important differences in the composition of air pollution in different cities and in different countries. They pointed out the difficulty of evaluating the effect of air pollution since the increase in the risk of lung cancer in cities was not greatly different from that seen in gas workers where the pollution levels were exceedingly high. The most likely explanation appeared to be that in each case the causative factor in the

great majority of cases was cigarette smoking and that small errors in estimating smoking levels obscured the true urban/rural gradient for lung cancer deaths. At about the same time Hitosugi (1968) reported large surveys from Japan which confirmed the dominant effect of cigarette smoking but which showed only a minor effect of air pollution among smokers and none at all amongst non-smokers.

It was evident at this time that there was a gradual swing away from the views expressed by Pybus (1963) to the view that lung cancer was a disease predominantly caused by cigarette smoking to which air pollution contributed little if any effect.

The relative contributions of cigarette smoke and air pollution to lung cancer mortality in the community were by now perceived as matters of the greatest importance. Because of conflicting evidence a number of important scientific bodies and eminent individuals published reviews in which the strength of the evidence for air pollution as a cause of lung cancer was reviewed.

For the purposes of this report the main findings of some of the most important of these documents will be presented.

2.1 Major Review Documents

(A) Biological Effects of Atmospheric Pollution.

Particulate Polycyclic Organic Matter.

Committee on Biological Effects of Atmospheric
Pollutants.

Division of Medical Sciences, National Research
Council.

National Academy of Sciences, Washington, DC 1972.

This document represented the findings of a major committee enquiring into the health effects of air pollution, specifically of particulate polycyclic organic matter on human health. It was noted that the size distribution of air pollution is fairly well known and in general consists of aerosols having exceedingly fine particle sizes. For example, 68% of the particulate pollution contributed by automobiles is less than 0.3 μ m in diameter. In contrast there is almost no information on the size distribution of particulate organic matter (POM). A list of polycyclic aromatic hydrocarbons which have so far been identified in urban air together with estimates of their carcinogenicity are shown in the table below.

TABLE #1

Polycyclic Aromatic Hydrocarbons

Compound	Carcinogenicity
Anthracene	-
C ₁₄ H ₁₀ m.p., 216° C; b.p., 340° C	
Benz[<i>a</i>]anthracene	+
Compound	Carcinogenicity
C ₁₈ H ₁₂ m.p., 158° C; sublimes (1,2-benzanthracene)	
7,12-Dimethylbenz[<i>a</i>]anthracene (9,10-dimethyl-1,2-benzanthracene)	++++
Dibenz[<i>a</i> , <i>j</i>]anthracene (1,2-7,8-dibenzanthracene)	+
.Dibenz[<i>a</i> , <i>h</i>]anthracene (1,2-5,6-dibenzanthracene)	+++
.Dibenz[<i>a</i> , <i>c</i>]anthracene (1,2-3,4-dibenzanthracene)	+
Phenanthrene	-
C ₁₄ H ₁₀ m.p., 101° C; b.p., 340° C	
Benzo[<i>c</i>]phenanthrene (3,4-benzphenanthrene)	+++
Fluorene	-
C ₁₃ H ₁₀ m.p., 116° C; b.p., 293° C	
Benzo[<i>a</i>]fluorene (1,2-benzfluorene)	-

Benzo[a]fluorene (2,3-benzfluorene)	-
Dibenzo[a,h]fluorene] (1,2-6,7-dibenzfluorene)	+
Dibenzo[a,g]fluorene (1,2-5,6-dibenzfluorene)	+
Benzo[c]fluorene (3,4-benzfluorene)	-
Dibenzo[a,c]fluorene (1,2-3,4-dibenzfluorene)	+

Compound	Carcinogenicity
Fluoranthene	-
C ₁₆ H ₁₀ m.p., 110° C; b.p., 393° C	
Benzo[b]fluoranthene (2,3-benzofluoranthene)	++
Benzo[j]fluoranthene (7,8-benzofluoranthene)	++
Benzo[k]fluoranthene (8,9-benzfluoranthene)	-
Benzo[ghi]fluoranthene	-
Aceanthrylene	-
C ₁₆ H ₁₂ m.p., 113° C	
Benz[j]laceanthrylene =cholanthrene	++
3-Methylcholanthrene	++++
Napthacene	
=benz[b]anthracene C ₁₈ H ₁₂ m.p., 341° C; sublimes	-

Naptho[2,1,8-qr]naphthacene
=naphtho[2,3-a]pyrene
(2',3'-naphtho-1,2-pyrene)

-

Pyrene

-

C₁₆H₁₀
m.p., 150° C; b.p., >360° C

Benzo[a]pyrene
(1,2-benzpyrene)
(3,4-benzpyrene*)

+++

Compound	Carcinogenicity
----------	-----------------

Benzo[e]pyrene
(4,5-benzpyrene)
(1,2-benzpyrene*)

-

Dibenzo[a,l]pyrene
(2,3-4,5-dibenzpyrene)
(1,2-3,4-dibenzpyrene*)

±

Dibenzo[a,h]pyrene
(1,2-6,7-dibenzpyrene)
(3,4-8,9-dibenzpyrene*)

+++

Dibenzo[a,i]pyrene
(2,3-6,7-dibenzpyrene)
(4,5-8,9-dibenzpyrene*)

+++

Dibenzo[cd,jk]pyrene
=anthranthrene

-

Indeno[1,2,3-cd]pyrene
(0-phenylenepyrene)

+

Chrysene

±

=1,2-benzophenanthrene
C₁₈H₁₂
m.p., 254° C; b.p., 448° C

Dibenzo[b,def]chrysene
=dibenzo[a,h]pyrene
(3,4-8,9-dibenzpyrene*)

++

Dibenzo[def,p]chrysene
=dibenzo[a,l]pyrene
(1,2-3,4-dibenzpyrene*)

+

Dibenzo[def,mno]chrysene
=anthanthrene
=dibenzo[cd,jk]pyrene

-

Perylene

C₂₀H₁₂
m.p., 273° C; b.p., ca.
500° C

Benzo[ghi]perylene

-

Compounds

Carcinogenicity

Coronene

-

C₂₄H₁₂
m.p., 438° C; b.p., 525° C

Aza-Arenes

Compounds

Carcinogenicity

Acridine

-

C₁₃H₉N
m.p., 111° C; b.p., >360° C

Dibenz[a,j]acridine
(1,2-7,8-dibenzacridine)

++

Dibenz[a,h]acridine
(1,2-5,6-dibenzacridine)

++

Dibenz[c,h]acridine
(3,4-5,6-dibenzacridine)

±

Carbazole

-

C₁₂H₉N

m.p., 246° C; b.p., 355° C

Benzofalcarbazole (1,2-benzcarbazole)	±
Dibenzofa,glcarbazole (1,2-5,6-dibenzcarbazole)	±
Dibenzofc,glcarbazole (3,4-5,6-dibenzcarbazole)	+++
Dibenzofa,ilcarbazole (1,2-7,8-dibenzcarbazole)	±

The most comprehensive information is available about benzo[alpyrene. There is a strong urban/rural gradient of about a factor of 10-fold and concentrations are also very much higher in cities during the winter in comparison with the summer months. A valuable summary of typical values of aerosol concentration for different geographic areas (annual averages) is presented as follows:

Typical Values of Aerosol Concentration for Different Geographic Areas (Annual Averages)

Location	N. particles/cm ³ _b	Mass Concen- tration ug/m ³ _c	Benzene- Soluble Fraction ofm. ug/m ³	Benzo[alpyrene Fraction of m. ng/m ³
Nonurban				
Continental				
General	103-104	20-80	1.1-2.2	-
California	103-104	39	2.8	0.48
Oregon	-	47	0.9	0.09
Colorado	102-104	14	1.1	0.11
Indiana	-	39	2.1	0.25
Maine	-	18	1.2	0.12
New York	-	29	1.8	0.25

So. Carolina	-	40	2.7	0.43
Maritime				
General	102-104	-	-	-
Pacific				
Offshore	102-104	19-146	1.5-6.1 ^d	-
Oahu, Hawaii	102-104	10-49 ^d	0.7-6.3 ^{d,e}	-
Urban				
Continental				
General	103-106	>100	7	-
Los Angeles	103-106	93	12.5 ^f	1.87
Portland	-	72	6.6	2.60
Denver	103-105	110	9.0	2.52
Minneapolis	103-105	70	6.1	1.18
Location	N. particles/cm ^{3b}	Mass Concen- tration ug/m ^{3c}	Benzene- Soluble Fraction of m. ug/m ³	Benzo[a]pyrene Fraction of m. ng/m ³
Chattanooga	-	105	6.9	4.18
New York	-	105	8.9	3.63
Greenville, S.C.	-	76	7.4	7.49
Maritime				
Honolulu, Hawaii	102-104	40	2.3	0.59
San Juan, Puerto Rico	-	77	6.9	1.42

The committee considered very carefully the use of benzo[a]pyrene as an index of air pollution and concluded that it correlated reasonably well with other POM. The arguments for using a single index is summarized as follows: "One of the common pollutants, benzo[a]pyrene, was chosen as an index of urban air pollution. Benzo[a]pyrene is taken as the primary index of air pollution, with the recognition that it is only one of the polycyclic organic materials in the air. The choice should not be taken to represent a conclusion that benzo[a]pyrene is the

causal agent in urban lung cancer. Its selection as an index is plausible for several reasons: It appears in solid form in air, is usually adsorbed on particles, and therefore can be filtered and collected. It is relatively easy to measure and is well correlated with other POM. It has been found to be carcinogenic in animals, and it is suspected of being carcinogenic in man."

In reviewing the strength of the evidence presented in various epidemiological studies it was noted that many were flawed because of poor estimates of pollution and lack of control of smoking levels. In general, an urban/rural gradient of lung cancer deaths in males is approximately 2-fold and the central difficulty is to estimate how much of this difference can be accounted for by differences in smoking habits. It was also evident that average concentrations of air pollution in the United States had fallen by a factor of at least 3-fold since 1950 and this added to the difficulties of interpretation.

Another problem which is of special significance to the present report on the health effects of POM in the mining industry in Ontario is the question of comparing the effects of BPA in the occupational environment with that associated with cigarette smoking. It appears that environmental exposure to British Gas Workers Doll et al. (1965) and Topside Coke Oven Workers Lloyd (1971) produces very much less lung cancer than the same amount of benzo[a]pyrene in cigarette smokers. This is one example where it is clearly very difficult to compare the effects of benzo[a]pyrene in different situations. The committee

summarized their conclusions as follows:

"It appears, then, that there is an "urban factor" in the pathogenesis of lung cancer in man. The polycyclic organic molecule mentioned most prominently in this report has been benzo[a]pyrene. It was felt that benzo[a]pyrene could be used as an indicator molecule of urban pollution, implying the presence of a number of other polycyclic organic materials of similar structure that may also have some carcinogenic activity. The standard measure of benzo[a]pyrene concentration in the air is the number of micrograms per 1,000 m³ of air. On the basis of epidemiologic data set against information regarding the benzo[a]pyrene content of the urban atmosphere, one can develop a working hypothesis that there is a causal relation between air pollution and the lung cancer death rate in which there is a 5% increase in death rate for each increment of urban air pollution. In this study, an increment of pollution corresponded to 1 ug of benzo[a]pyrene per 1,000 m³ of air. On the basis of this assumed relation, a reduction in urban air pollution equivalent to 4 benzo[a]pyrene units (i.e., from 6 ug/1,000 m³ to 2 ug/1,000 m³) might be expected to reduce the lung cancer death rate by 20%. These data, however, are not to be interpreted as indicating that benzo[a]pyrene is the causative agent for lung tumors. There is much to support the idea of synergism or cocarcinogenesis, especially with respect to cigarette smoking."

B. Review of Relationship Between Air Pollution and Lung Cancer.

Prepared by: Pike, M.C., Gordon, R.J., Anderson, B.E.,
Menck, H. R., and Soohoo, J.

This review was conducted under Contract #PH43-NCI-68-1030 within the Virus Cancer Program of the National Cancer Institute, National Institutes of Health, U.S. Public Health Service. Published in "Persons at High Risk of Cancer".

The authors refer to the large scale measurements of PAH in urban atmospheres undertaken during 1958-59, Sawicki et al. (1962). As an illustration of the change in concentrations over time they note that the median concentrations of BAP were 6 ug/m³ in the urban atmospheres but by 1969 this had fallen to less than 2 ug/m³. In the studies they considered that BAP correlated reasonably with total PAHs but varied according to the main source of the PAHs. BAP could contribute only a small portion of the total carcinogenic activity in air pollution and it therefore followed that its contribution to the total carcinogenicity would also vary according to the source of the total PAHs. Despite these difficulties they considered that it remained at present the best relative indicator of air pollution and of carcinogenic activity in the air of cities and could therefore be used to indicate decrements in activity over a period of time. However, it was clear that there were many other carcinogens present Gordon et al. (1973) and BAP could not

be regarded as a single causative factor.

In addition to the change in ambient levels of BAP that had occurred over time the authors drew attention to the change in particle size distribution that had occurred in these years. In the early years when BAP levels were high much of this was associated with coarse soot particles derived from the burning of fossil fuels. In more recent years this had changed so that the size distribution of the ambient pollutants was much finer. Unfortunately detailed information on these changes is not available and it is difficult to determine the effects that this might have on the exposed populations.

The authors also consider the evidence relating BAP levels in cigarette smoke and the occupational exposure in gas retort workers and in coke oven topside workers which had been reviewed by the National Academy of Science Report. In their view there had been errors in calculating the total effect of the occupational exposures due to the fact that exposures at work may be high but are only for a restricted part of the week. Recalculation, in their view, showed that the increase in cancer expected from occupational exposure was equivalent to that produced by urban air pollution. In their view an urban/rural gradient of about 2-fold in lung cancer rates was present and this was due to pollution. Cigarette smoking still appeared to have a very much higher response in relation to the amount of BAP inhaled. For all these reasons BAP remained a reasonably acceptable index of air pollution but certainly could not be

regarded as the single causative factor.

C. Atmospheric Pollution and Lung Cancer

Richard Doll. Environmental Health Perspectives,
Volume 22:23-31, 1978.

This report was prepared as part of a symposium on Air Pollution and Cancer which was held at the Karolinska Institute in Stockholm in March 1977.

Professor Doll addresses specifically the question of the urban/rural gradient of about 2-fold and considers whether this could be accounted for by air pollution or whether it is due to personal habits, mainly cigarette smoking. Emphasis is placed on the time scale of the epidemic of lung cancer which has occurred in industrialized countries. The increase occurred long after coal burning had reached its height and before the advent of significant diesel emissions. For these reasons it was quite clear that the epidemic as such could not be due to any great extent to either cause.

Amongst non-smokers it was also clear that there was little difference in urban or rural death rates and therefore atmospheric pollution appeared to have no measurable effect on this group. At present it appeared to be very difficult to be certain of the effect of atmospheric pollution. The data were compatible with no effect but an interaction between cigarette smoking and air pollution could not be excluded.

He concluded as follows:

"The ambient atmosphere, particularly in towns, contains a variety of substances that are capable of causing cancer of the lung in men who are exposed to large amounts of them in the course of their occupation. General knowledge of carcinogenesis suggests that at low doses the carcinogenic effect is likely to be linearly proportional to the dose received. It should, therefore, be assumed that the small amounts present in the atmosphere contribute to the causation of some of the cases of lung cancer that occur in clinical practice.

Observations on men indicate that by far the most important cause of lung cancer is cigarette smoking and that other factors characteristic of town air have very little effect, except in so far as some of them may interact with it. Extrapolation from occupational studies suggests that this effect is unlikely to be large. In the absence of cigarette smoking, the combined effects of all such atmospheric agents cannot be responsible for more than about five cases of lung cancer per 100,000 persons per year in European populations."

D. General Air Pollution and Cancer in the United States.

Hammond, E.C., Garfinkel, L.

Preventive Medicine 1980;9:206-211.

This report concluded as follows:

"In a review of the literature published some years ago

Cornfield et al. (1959) the authors concluded that there was no firm evidence to support the hypothesis that general urban air pollution increases the risk of lung cancer to an important degree, if at all. Data from our study support that conclusion and we are unaware of any evidence which convincingly leads to a contrary conclusion.

Available evidence does not rule out the possibility that general urban air pollution may perhaps lead to a slight increase in the risk of lung cancer."

3. BIOLOGICAL EVALUATION OF THE CARCINOGENIC POTENTIAL OF AIR POLLUTION - THE AMES TEST.

The previous sections have outlined developments in the evaluation of the health effects of air pollution in terms of the concentrations of specific PAHs. It has been emphasized that the selection of any one chemical compound cannot be used as an indicator of the total carcinogenic potential of any given atmosphere. Because of this problem there are obvious attractions in attempting to measure the potential health effects by some more direct approach. Long term animal exposures might be one method but a more convenient and practical test would be the microbial assays developed by Ames et al. (1975). Many tests of this type have now been produced which measure mutagenic changes in various types of biological cell with the implication that these also relate to carcinogenic potential. However, there are a number of problems associated with this approach. As Wei et al. (1980) emphasized it is first necessary to remove cytotoxic materials from the sample to be tested since these toxins may generate false negative results.

Unfortunately even when the tests are undertaken correctly it is not possible to compare the carcinogenic potential of one atmosphere with that of emissions from another source because of qualitative differences in the mutagens in the different mixtures. The various types of Ames Test also respond in a different manner to the various chemicals. For example, salmonella tester strains have bacterial enzymes which can

activate some chemicals to reactive intermediates while other cells do not have this property. As a result, the Ames Test is especially sensitive for detecting direct-acting mutagens which contain nitrogroups because the nitrogroup is activated by nitroreductases in the salmonella McCann and Ames (1976). Important implications can be derived from direct-acting Ames Test experiments (without the addition of activating enzymes from liver homogenates as distinct from indirect-acting results where the liver homogenates must be added in order to obtain positive results). For example diesel exhaust samples examined by the authors generally behave as direct-acting mutagens. On the other hand they emphasize that unsubstituted PAH, such as benzo[a]pyrene (BAP) are indirect-acting mutagens Kier et al. (1974). This is an example which demonstrates that the principle mutagens in diesel exhaust must therefore be different from the mutagens present in coke oven emissions and in tobacco tar. Consequently, the authors believe that the toxic properties of one mixture cannot be related to, or predicted from, measurements carried out on pollution of other origins.

In a critical review of mutagenic tests in the assessment of diesel exhausts Rosenkranz (1982) drew attention to a number of issues. Chemical analyses as well as microbiol mutagenicity assays have suggested that 1-nitropyrene is the major mutagenic species accounting for approximately 20% of the mutagenic activity present in diesel emissions. The other direct-acting mutagenic chemicals identified in diesel emissions cannot

account for the remaining 80% of the mutagenicity of diesel emission material on the basis of their occurrence. It appears likely that 1,8-dinitropyrene is present in very low concentrations which might not be routinely detectable chemically. However, this is probably the most potent mutagenic chemical reported in the literature and could easily account for the remaining 80% of the observed mutagenic potencies of diesel emission materials. The author goes on to summarize that the situation which has led to acceptance of benzo[*a*]pyrene, rather than mutagenic species such as nitropyrenes, as the surrogate for setting clean-air standards (U.S. Congress, 1977; U.S. Environmental Protection Agency, 1979a, b) derives from a philosophical consideration that the chemicals that are most abundant in the environment should be the ones that should be targeted for control (mass-oriented approach). On the other hand, an approach directed at elimination from the environment of the biologically active species, even though they might be present in minute quantities, might have led to the recognition of the nitropyrenes earlier. Indeed, nitropyrenes have been recognized in the environment as a result of such an activity-oriented search: e.e., the identification and subsequent reduction of nitropyrenes from carbon black (Rosenkranz et al., 1980).

4. COMMENTARY ON REVIEW DOCUMENTS

This review has selected a few of the major studies which have been published in relation to atmospheric carcinogens and the development of lung cancer in the exposed populations. Its purpose has been to present an overview survey of the very considerable literature which has now accumulated and to illustrate the profound change in thinking which has occurred during the last 50 years. From a position whereby air pollution was believed to be the major determinant of lung cancer and which could be evaluated by the analysis of benzo[*a*]pyrene in the atmosphere, current epidemiological evidence no longer supports these views. Not only is cigarette smoking clearly perceived as being the major cause of lung cancer but considerable doubt is now expressed of the proposal that air pollution contributes any significant effect. Moreover, it is evident that the choice of benzo[*a*]pyrene as an index of air pollution can serve only to indicate relative changes in the biological effects of pollution derived from a single source. It is evident that it can no longer be used as an index of the total carcinogenic potential of an atmosphere and most certainly cannot be used for comparing one atmosphere with another. Not only are the overall composition of such atmospheres very different but also they are associated with particulate aerosols of very different size distributions and it is highly likely that these influence the biological effects.

It is also evident that the relationship between air pollution and lung cancer remains highly controversial and it is not improbable that no final and universally agreed solution will evolve. This is because of the long latency in the development of malignant disease following initial exposure and because air pollution levels and smoking habits have changed so dramatically during the last 20 years. Changes in cigarette smoking involve not only the numbers of cigarettes smoked but also their tar composition and the influence of filter cigarettes. Air pollution has also changed in concentration, composition, origin and in the size distribution of associated particulate matter.

5. POLYNUCLEAR AROMATIC HYDROCARBONS IN ONTARIO MINES

5.1 INTRODUCTION

In this section the methods of sampling and analysis of Polynuclear Aromatic Hydrocarbons (PAHs) used by Westaway in his investigations Westaway (1980); Westaway (1981); Westaway (1982); and Westaway (1983) are reviewed. Taking due account of the issues discussed in preceding sections the data of the Westaway reports is then used to derive the "best" exposure estimates of the underground miners to the Benzo[a]pyrene (BAP). These exposure estimates of the Ontario Underground Nickel, Uranium and Salt miners are then compared to the B(a)P exposures reported in other industrial work environment and in the ambient (outdoor) air. This is intended to show where the Ontario Miner's exposure lies in the spectrum that ranges from outdoor air to highly B(a)P polluted work environment.

5.2 SAMPLING METHODS

AIR SAMPLING FOR PAHs

High Volume sampling and low volume (personal) sampling are the two main methods of collection of PAHs in the air.

High Volume (Hi-Vol) Sampling

In this method airborne material is collected on an organic binder free glass fibre filter paper by the use of high volume

sampler or high suction source. Most of the Hi-Vol samples are collected on a 8" x 10" filter paper at a flow rate of 1 to 1.7 m³/min (1000-1700 l.p.m. = 35 to 60 c.f.m.). Almost all the outdoor air samples are collected in this manner, since the very low concentration of PAHs requires much longer (24 hrs and longer) collection at this rate in order to obtain sufficient sample for an accurate analysis.

This Hi-Vol method was also utilized extensively in the past in the investigation of PAHs in workplaces such as gas works, coke plant, Iron and Steel Industries, Foundries, etc.

A few investigations have used a different filter size and different flow rate in their Hi-Vol sampling of PAHs in workplaces. Hi-Vol can only be used as an area/location sampler and is not suitable for personal sampling.

Low Volume (Personal) Sampling

This recent development is essentially a personal sampling technique which requires collecting airborne material by a portable personal sampling pump at a flow rate of about 2 l.p.m. The sampling media generally consists of a fibreglass pre-filter with a silver membrane as the backup filter. The common set up in use is a 37 mm diameter 99.9% efficient fibreglass filter paper with a 37 mm diameter 0.8 um pore size silver membrane filter. Since the portable sampling pumps have only become available during the early 1970s, the earlier investigators have generally relied on the other method (Hi-Vol). The low volume

(personal) sampling offers a distinct advantage over the Hi-Vol method since it permits the collection of personal samples (a more accurate estimate of worker's exposure) as well as the area/location samples, while Hi Vol can only be used to obtain area/location samples.

Although low volume sampling is much more attractive for health exposure assessment, it can present significant problems where the exposure PAHs is very low. In such cases several personal samples need to be taken and pooled to obtain exposure estimates Gibson et al. (1977), Verma et al. (1982). Although time consuming it provides a more representative worker's exposure estimate.

Although it is well known that the Hi-Vol sampler generally collects larger particles of airborne dust than low volume (personal) samples, in cases where the PAHs are at very low levels Hi-Vol remain the only feasible method.

Since the aims of the Westaway studies were to test out various engineering hypothesis (e.g. effects of catalytic converts on production of PAHs, effects of various fuels on PAHs) the selection of Hi-Vol sampling was a reasonable and an acceptable approach. If the major aim of the studies were to assess the full shift PAHs exposure of the underground miners then an appropriate sampling strategy aimed to obtain such information by Hi-Vol or low vol sampling would be required.

Westaway (1982) has reported that the Hi-Vol method in sampling diesel underground has collection efficiency of less

than 60% and possibly only 20%. This implies that a large amount of PAHs are not collected by this method. Since this finding is based on only a limited number of samples and experience in one work setting (Diesel underground), further confirmation is required based on evidence derived from more samples and evidence coming from other industrial Hi-Vol sampling. The factors which need to be examined include effects of flow rates, temperature, humidity and composition of other contaminants in the workplace.

5.3 ANALYTICAL METHODS

The analytical techniques employed for PAHs can be broadly classified in three groups as:

- i) indirect gravimetric estimates by benzene soluble/cyclohexane or other solvent soluble/extraction
- ii) instrumental methods primarily relying on retention time and possible spectrophotometric identification, e.g. gas chromatography, column chromatography and spectrophotometry, thin layer chromatography and high pressure liquid chromatography.
- iii) the method that unequivocally identifies and quantifies individual PAHs, e.g. gas chromatograph-mass spectroscopy (GCMS)

The method used by Westaway in his studies is from the second group - thin layer chromatography (TLC). The method was

developed and tested extensively in the laboratory as well as on underground samples. The procedure was tested for important parameters e.g. most suitable solvent for extraction, separation of 13 individual PAHs, effect of storage on the samples' integrity, and the photodecomposition of PAHs.

In his reports Westaway has described several laboratory experimental validation procedures and the precautions that were taken to optimize analysis in the laboratory. They are good and therefore, the results should be an accurate estimate of the PAHs present in the analyzed samples.

5.4 EXPOSURE ESTIMATES

It is very difficult to derive an accurate estimate of full shift exposure of the underground miners to the PAHs from Westaway's data. There are several reasons for this. The most important is the design of the study.

Since the study was designed to test out various engineering control hypothesis (e.g. Does a catalytic converter reduce PAHs? Is one fuel better than another for the production of PAHs?), the sampling and analysis scheme is inappropriate for the exposure estimation. In attempting to estimate worker exposure it is important to consider not only the site but also the derivation of sampling. The last point is of special relevance when it is required to estimate the total amount of PAH inhaled by a miner. Levels may fluctuate considerably during the shift

and full shift sampling is, in general, the best way of obtaining a reliable estimate. Full shift personal sampling takes account of the movement of the miner from one work station to another, the variability of operations at a given work station and the travelling time to the work station.

The next important point that is relevant is the fact that many observations and conclusions are based on too few samples to be reliable. It is possible however, to get an estimate of some specific PAHs (e.g. B(a)P) by using all available data from the Westaway's reports. The limitations of using B(a)P as an index of carcinogenicity have been discussed in earlier sections of this report.

It was, therefore, decided to tabulate all B(a)P measurements from the Westaway reports irrespective of where and for what purposes they were taken. The low B(a)P data on fresh air raises as well as high B(a)P data generated during testing of catalytic converters were all included.

All B(a)P data was essentially segregated by mine types as nickel, uranium and salt. It was assumed that each individual B(a)P measurement represented a valid sample of the distribution of underlying B(a)P concentrations. Since a miner has to travel in fresh air as well as work in raises and stopes it is not unreasonable to include all data points for exposure estimation. Available data were also gathered for outdoor air. In table #2 raw data on nickel, uranium and salt mines are shown. Also included in the table at the bottom is the descriptive statistics

which were computed based on the grouped data. The data appears to be log normally distributed. There was no evidence to suggest log normal lack of fit based on χ^2 test. The respective geometric means could then be taken as to represent the best estimates of exposures to B(a)P to the underground miners as follows:

Nickel Miners - 0.64 ng/m³

Uranium - 1.96 ng/m³

Salt - 0.43 ng/m³

The possible health implications of these concentrations are reviewed in the following section of the report.

6. AN APPROACH TO THE EVALUATION OF THE HEALTH EFFECTS OF PAHs IN ONTARIO MINES

The purpose of this report has been to evaluate the ambient levels of PAHs in Ontario mines reported by Westaway and Foulds (1982). The first chapters have shown the current information on the health effects of PAHs has been derived from large scale surveys of urban air pollution and from much more limited data obtained in the occupational environment. For a number of reasons it has been convenient in such surveys to use BAP as an index of the carcinogenicity of air pollution. However, it gradually became evident that such a simplification had only limited utility. In general the following statements describe the consensus of current thought with reasonable accuracy.

1. Current levels of air pollution in industrial cities of North America are unlikely to be significant causes of lung cancer.
2. BAP is of limited utility as an index of the carcinogenicity of such atmospheres.
3. BAP, other PAHs or combinations of such do not serve as measures of carcinogenicity and cannot reliably be used as predictors of such in a given atmosphere by making use of epidemiological evidence derived from other sources.
4. Biological assays of carcinogenicity of the Ames type also have similar limitations. As a result of

mutagenic contributions from the many different PAHs it is not helpful to estimate the biological activity of mine atmospheres, to compare it with that of urban pollution and then to infer the dimensions of health hazards within the mining environment on the basis of epidemiological surveys of city dwellers.

These uncertainties place severe restrictions on the information that can usefully be derived from sampling data in the mining environment. Most certainly the evidence that diesel emissions contain mutagenic substances immediately indicates that controls should be considered to reduce worker exposure. It is much more difficult to rate the priority of such action in comparison with other hazards and to give some idea as to how rigorous the control program should be. The desirability of an environment free of mutagenic co-carcinogenic substances cannot be disputed. However, the fact that pollution in cities has probably had little, possibly no, impact on lung cancer rates while at the same time certainly containing BAP and other carcinogens indicates that the presence of such substances does not automatically imply that there is a major health hazard. This is an important point for miners. It is facile to equate a positive Ames Test on an environment or the presence of BAP with a real health risk without some attempt to quantify its magnitude. It is even more important to consider the consequences of inaction while awaiting the results of adequate long-term epidemiological data. The history of health in industry is

already more than replete with examples of the futility of demonstrating lethal health risks at a point in time when it is too late to help affected workers.

A number of such epidemiological surveys are in progress but an evaluation of these is not within the terms of reference of the present report. To date, initial results have not identified a significant lung cancer risk and it is possible that a long-term follow-up will confirm these findings. On the other hand such an eventuality cannot be relied upon and it is necessary to make the best possible evaluation on the basis of available information.

The validity of the measurement techniques in the Westaway Report , Westaway and Faulds (1982) was reviewed in Chapter 5. The problems of making evaluations of the health hazard that might be associated with the PAH levels reported has been clearly identified in the present report. Despite these restrictions some tentative conclusions can be presented.

Firstly, it is helpful to begin by considering current levels of air pollution in cities. The levels of B(a)P in outdoor air of Sudbury has been tabulated in Table 3 along with the concentrations found in other cities. Descriptive statistics similar to the ones for underground mines have been calculated and included in the table.

The B(a)P concentrations of Sudbury air and the underground nickel, salt and uranium mines can be viewed in relation to other work environments. Lindstedt and Sollenberg (1982) in

their review of polycyclic aromatic hydrocarbon in the occupational environment, based on health effects (mainly lung cancer) and the exposure data, have suggested high to low risk categories for various workplaces based on the level of B(a)P exposure. Their categorization is reproduced in Table #4. The Ontario mines would fall in his lowest hazard category #5.

The underground miners B(a)P exposure estimates can be placed in proper perspective when it is compared with the estimates of the exposure in other workplaces along with the levels that are found in the outside air. Such a comparison is shown in the Table #5. This is important since it places Ontario miners exposure in relative perspective.

The importance of the B(a)P levels found in cities is that they almost certainly refer to pollution concentrations which do not cause a measurable lung cancer risk. The uncertainties over the two-fold urban/rural gradient in lung cancer deaths was discussed in earlier sections of this report. Even if the gradient is real and is attributable to air pollution it is certain that this was the result of exposure in earlier years to levels greatly in excess by Katz et al (1978).

A first step in the analysis is thus to compare the measurements obtained underground by Westaway et al. (1982). In making such comparison attention has been drawn to the problem that the sampling strategies used were designed for engineering purposes and not in order to estimate exposure to miners. For example the authors comment on the very high PAH levels measured

in the proximity of drilling operations, although, in fact the data of the single sample reported in Table II of Westaway (1982) does not appear to be very different from the overall mean of samples of diesel origin. In addition, they state that much of this was likely to be absorbed on oil droplets which were of a size which was greater than that which could be inhaled. This is an interesting possibility, but we know of no data on the size distribution of such particulates and this would merit further investigation.

If the most carcinogenic compound (BAP) is selected as an index it is evident that the mean concentration is about that of the Sudbury air in 2 mines, (nickel and salt mine 2) but considerably higher in the others. Even at the highest however (3.75 ng/m³ in the uranium mine) the level is below that of the average U.S. urban concentration reported in 1968, Sawicki et al. (1965) and approximately equal to that of the city of Hamilton (site #29025) during the months October to December 1975 (Katz et al. 1978). The levels may be compared with that found in Finnish mines (Hakela et al) who found concentrations of BAP varying from 6 to 36 ng/m³. Unfortunately their report does not indicate which were derived from personal samples and which were from high volume stationary samples. For this reason it is difficult to evaluate the data or to use it in relation to lung cancer mortality in Finnish mines.

In comparing these mean city levels with those in a mine it must not be forgotten that underground exposure is limited to

40 hours per week. Urban residents inhale their atmospheres for 168 hours per week. A fair comparison which allowed for lower week-end city air pollution levels might be to multiply the urban levels by a factor of 2 in order to produce an equivalent health impact.

On this evidence it appeared that the mining environments presented by Westaway and Faulds(1982) are of the order of air pollution experienced by city dwellers - being greater than those experienced by residents of Sudbury but probably less than those of Hamilton in 1975 or of the average U.S. city dweller in 1968. The evidence relating these environments to lung cancer increasingly suggests that the effect is small if present at all. The evidence thus suggests that the underground PAHs are undesirable, that efforts should be made to reduce them but that the health impact as far as lung cancer is concerned is likely to be small.

The Westaway and Faulds (1982) report reemphasizes the point made many years ago that much of the mutagenic activity of diesel exhaust is likely to be present in compounds other than BAP. The most important species is likely to turn out to be the nitrated PAHs (nitro PAHs). For this reason the authors undertook a sampling program with evaluation of the carcinogenic potential of the mine atmosphere by means of bacterial assays. As might have been anticipated the results were strongly positive. For the reasons which have been discussed earlier in this report we are unable to quantify this effect in terms of the likely health

impact on exposed miners. Neither can we indicate appropriate action other than the general observation that the presence of potential carcinogens is undesirable and should be reduced when ever possible.

On a more practical level it is of interest to examine the implied health effects of adding filters and/or catalytic converters to diesel powered engines. The hypothesis is that particulate matter i.e. carbon is readily trapped by filters and that, since PAHs are thought to be adsorbed on the particles, that this should lower the PAH and also nitro PAH levels in the mine.

The authors therefore evaluated the effect of adding a USBM filter to the exhaust system of Deutz diesel. To their surprise they found little difference in the concentrations of PAHs downstream of the motor and concluded that a filter which removed diesel particulates could not be assumed to remove PAHs. The reasons for this failure to trap PAHs are not known but it appears to the present writers that the temperature of the filters might have been very high and that, under such circumstances, PAHs in the vapour phase would not be condensed on the carbon material accumulating in the filter. The importance of temperature in determining the collection efficiency for PAH's on carbon particulates which have been trapped in filters is well recognized and the EPA have specified a maximum allowable sample temperature of 52° C. No data on the temperature of the filter apparatus is cited in the Westaway

report. Even if this mechanism was not the cause of the low collection efficiency for PAH's an alternative explanation could account for the finding. The mechanical vibration associated with the mining environment has been postulated as causing resuspension of particulate matter so that it can enter the mine atmosphere (Mogen, personal communication 1986).

Westaway and Faulds (1982) even extend this discussion to suggest that the lung cancer risk following the addition of the filter might be greater since the escaping PAHs would not be able to condense on particulate matter and that this would result in aerosols having a greater proportion of respirable particles (note the term respirable is a misnomer in this context, the word inhalable is more appropriate). We believe that, in the absence of information concerning the size distribution of the PAHs or nitro PAHs with or without a filter that such speculations are inappropriate. For example if the size of PAH particulates were exceedingly small (say less than 0.01 μm dia.) then the increased deposition of particles in the nose or mouth as a result of diffusion could reduce particle penetration to the major bronchia. Clearly some experimental work to measure the size of the PAH particles must precede any meaningful discussion. We also take exception to the comments concerning the reputed greater risk of smoking cigarettes with filters (the reference for this remarkable information is not given). Discussion in the report concerning particle penetrance to the alveoli does not give an appropriate description of the

mechanisms of carcinoma of the bronchus since alveolar deposition is not relevant in the pathogenesis of tumours of the large airways. Moreover, the current gradual decline of the death rates from lung cancer in the male population of North America is usually attributed to lower tar yields but also to the use of filter cigarettes. In general, it would be in accordance with current thought to suggest that the addition of filters to diesel exhausts certainly removes much particulate matter and, for this reason, should be perceived as being a beneficial factor. The finding that this does not lower PAH levels should not be suggested as leading to unexpectedly greater health risk without some fairly solid support. The suggestion is interesting and merits research but, in our view, is unlikely to be substantiated. In practice this aspect of the effects of adding a particulate filter (the USBM filter) has less relevance following the introduction of the CORNING filter. This appears to produce substantial reductions in the concentrations of all PAH's (Laurentian 1984-85-86 Contract; Mogen, personal communication 1986) and it would demand very strong evidence from arguments based on postulated changes in particle size distribution to support this that this reduction would not be associated with reductions in the risk to health.

The effects of adding a PTX or Dieseler III catalyst converter are most interesting. These are designed to lower CO levels by conversion to CO₂. The finding that this could result in higher PAH levels and greater mutagenic activity certainly

suggests that the introduction of converters should be considered carefully. They are unlikely to be beneficial since CO concentrations are not, in general, a health problem. We found some difficulty in evaluating the effects of the catalytic converters from the report because of variability in the results obtained on different occasions. These technical problems illustrate the difficulties of undertaking research in the field since more than one variable is likely to be changed on each occasion. The development of catalytic converters appears to be proceeding rapidly. For example, a recent report (Mogen et al. 1985) describes the use of a catalyzed wire mesh trap. This produced significant reductions in total particle emissions from heavy-duty diesel engines, in the level of PAH's and, most importantly, in Ames mutagenic activity. It seems that the increased mutagenic activity found by Westaway when using a PTX or Dieseler III converter may refer only to those models.

A number of interesting issues can be identified in this research report. Two methodological issues merit special attention. Firstly, it is clear that there are difficulties in assessing the effects of a single variable such as the presence or absence of a catalytic converter when the experiments are carried out in the mining environment. There are real benefits to be obtained if a study is carried out under carefully controlled conditions such as an experimental stope rather than under normal working practice.

Secondly, it will be evident that the sampling strategy was

carried out from an engineering viewpoint. In these circumstances it is exceedingly difficult to interpret results and to evaluate possible health hazards. For health assessments it is necessary to estimate exposure in relation to workers using time weighted averages and to use appropriate size selective sampling devices. The situation is of course often reversed and engineers have equal difficulty in interpreting environmental data when these were obtained for health surveillance purposes. Probably a joint team approach is required to resolve this paradox.

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Table #2

Airborne Concentration of B(a)P in the Mines in ng/m³

Nickel Mines

84., 4.6, 3.2

0.2, 1.4, 0.3, 0.8, 0.5, 1.3, 0.54, 0.59

0.39, 1.2, 0.4, 1.2, 0.86, 0.57, 0.79, 0.43, 1.6,

0.03, 0.06, 0.10, 0.06, 0.51, 0.13, 1.6,

0.5, 0.31, 1.63, 5.4, 0.76, 1.6,

0.2, 0.1, 0.74, 0.12, 0.28, 0.65, 0.08

0.03, 0.03, 0.07, 3.51, 0.14, 0.54, 0.60

0.08, 0.38, 0.06, 0.17, 0.02, 0.02, 0.01,

1.31, 0.29, 0.60, 1.2, 0.31, 0.39, 0.39,

1.02, 1.60, 0.89, 2.21, 0.98, 1.04, 0.75

0.38, 0.29, 0.34, 1.63, 1.24, 2.22, 0.98

1.64, 3.02, 3.42, 1.60, 5.22, 4.36, 6.49

0.14

Uranium Mines

1.9, 3.9, 0.4,

2.1

1.41, 3.9, 3.75, 2.65, 3.50, 2.50,

Reference

Westaway, (1980)

page 51

Westaway (1981)

pages 11, 14, 39, 40

48, 51, 60

Westaway, (1982)

pages 6, 7, 23, 30

32, 45, 53, 62, 65,

82, 106, 114

Westaway (1983)

page 16

Westaway (1980) p.50

Westaway (1981) p.55

Westaway (1982)

page 6, 53, 106

Salt Mines

.006, 0.54, 0.28, 0.16, 0.38, 0.19, 0.1,
0.58

Westaway (1981)

page 33,55

1.08, 2.30, 1.67, 1.04, 0.42, 1.69, 0.58
1.36, 0.58, 1.63

Westaway (1982)

page 50,53,58,106

SUMMARY DESCRIPTIVE STATISTICS

MINE TYPE	NUMBER OF OBSERVATION	MEAN			RANGE	
		ARITHMETIC	GEOMETRIC	GEO.STD.DEV.	MIN	MAX
Nickel	83	1.10	0.64	2.2	0.01	8.4
Uranium	10	2.40	1.96	2.3	0.4	3.9
Salt	18	0.66	0.43	2.5	.006	2.3

TABLE #3

Airborne Concentration of B(a)P in Outdoor Air in ng/m³

Canada

Sudbury

Source

0.17, 0.11, 0.34, 0.44

Katz et al (1978)

0.02, 0.02, 0.38, 0.11, 0.26, 0.37, 0.25

Westaway (1982)

1.41, 0.37, 0.15, 1.31, 0.02,

page 6,12,14,19,25

Other Cities in Ontario (Toronto, Hamilton, Sarnia)

0.79, 1.04, 1.7, 0.72, 0.66, 0.41

Katz et al (1978)

0.73, 0.81, 1.4, 2.35, 3.50, 1.93, 0.34,

0.11, 0.60, 0.19

U.S.A.

Average U.S. Urban 5.7

Katz et al (1978)

New York 1.15, 1.30

Summary Descriptive Statistics

Cities	Number of Observations	Mean			Range Min - Max
		Arithmetic	Geometric	Geo.Std.Dev.	
Sudbury	16	.28	.15	2.2	0.02-1.4
Other Ontario	16	.81	.53	2.1	0.11-3.5
Cities					

TABLE #4

Categorization of Workplaces Based on B(a)P

[Reproduced from Lindstedt and Sollenberg (1982)]

1. Very high B(a)P exposures (10 ug/m³)

Gas and coke works (topside work)

Aluminum works (some jobs, eg. pin setters, potmen, cranimen, etc)

Manufacturing of carbon electrodes (pitch bin workers, etc.)

Handling of molten tar or pitch (roofing, paving, insulation, coating, etc.)

Chimney sweeping (from top)

Asphalting if asphalt is mixed with tar (some jobs)

2. Fairly high B(a)P exposure (1-10 ug/m³)

Gas and coke works in general (nontopside work)

Blast furnaces

Steel works (some jobs)

Manufacturing of carbon electrodes (in general)

Aluminum works (in general)

Asphalting if asphalt is mixed with tar (in general)

3. Moderate B(a)P exposure (0.1-1 ug/m³)

Steel works (in general)

Foundries (some jobs)

Welding of rails on track

Manufacturing of soderberg electrode paste

4. Low B(a)P exposure (0.01-0.1 ug/m³)

Automobile repair shops

Asphalt manufacturing (from petroleum)

Foundries (in general)

Construction of tunnels and rock chambers

Aluminum electrolysis hall with prebaked electrodes

5. Very low B(a)P exposure (0.01 ug/m³)

Iron mines

Garages

TABLE #5

Range of Concentrations of B(a)P in Occupational Investigations, Ontario Mines, and in the Outdoor Air in ($\mu\text{g}/\text{m}^3$) Modified and Expanded from Verma et al (1982)

Source	Operations	Type of sample/ flow rate	Range Min-Max
Commins et al (1957)	Merton bus garage	Area 1 m^3/min	.003-0.47
Lawther et al (1965)	Gas work retort house	Area 1.53-3. 1.p.m.	n.a.-2330
Tanimura (1968)	Iron and steel works	Area .2-.5 m^3/min	.040-4.17
Kreyberg (1959)	Industrial plants	Area 1 m^3/min	.180-7.30
Sawicki et al (1962)	Roof tarring	Area 1.4 m^3/min	.001-78.0
Masek (1971)	Coal & pitch coking plants		
	Lazy (1963-1967)	Area 25-30 1.p.m.	.020-296.23
	Ostrava (1966-1967)	Area	.050-33.28
	Vitezny (1966)	Area	.020-94.80
	East Slovak	Area	.310-159.00
Jackson et al (1974)	Coke oven	Area 1.4-1.7 m^3/min	.025-15.9
Schimberg et al (1980)	Iron Foundries	Area	n.d.-57.5
Bjorseth et al (1978a)	I. Aluminum reduction plant	Area 17 1.p.m. Personal 2 1.p.m.	n.d.-9.0 0.800-116.3

Bjorseth et al (1978b)	II. Coke plants	Area 17 1.p.m.	8.000-135.00
		Personal 2 1.p.m.	.0.500-43.18

Gibson et al (1977)	Steel Foundry	Personal 2 1.p.m.	0.020-0.30
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Verma et al (1982)	Iron & Steel Foundries	Area 2 1.p.m.	n.d.-8.05
		Personal 2 1.p.m.	

Westaway (1980,	Nickel mines	Area-Hi-Vol	.00001-.0084
1981, 1982, 1983)	Uranium mines	1.4-1.7 m ³ /min	.00004-.0039
	Salt mine		.000006-.0023

	Outdoor Air		
Katz (1978)	Sudbury	Area-Hi-Vol	.00002-.0014
Westaway (1982)	Sudbury	1.4-1.7 m ³ /min	
Katz (1978)	Outdoor Air - Other	Area Hi-Vol	.00011-.0035
	Canadian Cities	1.4-1.7 m ³ /min	
Katz (1978)	Outdoor Air, U.S.A.	Area Hi-Vol	
	Average Urban	1.4-1.7 m ³ /min	.0057
	New York		.00115, .00130

na = not available nd = non-detectable